**Title**
Evaluation of the Change in Eruption Angulation of Canines and Premolars after Phase I Expansion



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Evaluation of the Change in Eruption Angulation of Canines and Premolars after Phase I Expansion

by
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**Evaluation of the Change in Eruption Angulation of Canines and Premolars after Phase I Expansion**

**Lauren Frisch**

## Abstract

Objectives: The aim of this study was to evaluate the effect of maxillary rapid palatal expansion on the eruption vector of the canines and premolars in the early mixed dentition using CBCT.

Methods: The study population consisted of 42 individuals 7-11 years of age in the early mixed dentition, with all Ds and Es present, and all permanent first molars, maxillary central incisors and lower incisors erupted. No subjects had posterior crossbites or severe sagittal discrepancies. The treatment group of this study consisted of 21 children who had rapid palatal expansion using a Hyrax maxillary expander and active lower lingual arch in the early mixed dentition, as well as phase II comprehensive treatment records. The control group of this study consisted of 21 subjects who did not undergo rapid palatal expansion following their phase I records but returned in the full permanent dentition for comprehensive treatment records. All subjects were imaged using cone beam computed tomography (CBCT) as a part of their initial records, and again prior to beginning comprehensive Phase II treatment in the full permanent dentition.

Results: A canonical variate analysis revealed significant differences between control and treatment groups ($p=0.03$). There were no significant differences between groups in a principal component analysis, however mild differences between principal component 2 and principal component 8 were observed. Principal component 2 was responsible for 14% of shape variation and was the only principal component in this study to demonstrate shape differences between the treatment and control groups. Canines and premolars erupted with a slightly more upright angulation in patients treated with phase I expansion than those who were untreated.

Conclusions: Canines and premolars were slightly more upright in patients who had phase I expansion than patients who did not. These differences were very minor and were statistically significant in a canonical variate analysis (p=0.03).

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## Background

### *Transverse Skeletal Constriction*

The presence of a transverse skeletal deficiency, which constricts the dental arch, affects the ability of the permanent teeth to erupt into ideal, upright positions.[1,2] Posterior crossbites are primary clinical indicators of maxillary transverse skeletal discrepancies and affect 8-23% of the general population [2–4]. Midline deviations, mandibular shifts on closure, crowding, dental arch distortion, and anterior interferences resulting in abrasion may also indicate that posterior transverse discrepancies are present [5]. Patients who do not present with these distinctive dental clinical indicators may still possess skeletal constrictions that, although may be more challenging to diagnose and measure, would benefit from orthopedic treatment. One study found that 32% of subjects without posterior crossbites still had a palatal base equally as narrow as patients with unilateral crossbites. In these patients, skeletal transverse constrictions were only camouflaged by dental compensations of 'superior convergent' maxillary molars that are tipped buccally to maintain positive overjet [6]. Even without posterior crossbites, these patients may be at a high risk for developing some of the same downstream problems as patients with unilateral crossbites.

### *Expansion Therapy*

Transverse skeletal constrictions of the maxilla are of clinical concern because they can lead to unfavorable dental eruption and occlusion, asymmetric skeletal growth of the jaw and TMJ [2,4,7], and even upper airway constriction [8]. Early treatment is indicated in patients who may be at risk for these downstream effects. The most common treatment for transverse skeletal constriction in the mixed dentition is rapid palatal expansion [9,10]. Maxillary expansion to correct unilateral posterior crossbites is stable in 84% of patients, preventing these problems from worsening in the permanent dentition [2,11]. There are many different designs for palatal expanders. Some expanders, including the Haas, Hyrax, and quad helix, are fixed in place with bands around the posterior teeth, typically the first molars. Other expanders, like the Schwartz, are removable to allow the patients to brush their teeth more easily. The obvious drawback of a removable expander is that its success is dependent on patient compliance, whereas the fixed expanders, especially the Haas which has acrylic shelves that sit against the palate, are associated with worse oral hygiene, as they are more difficult to clean. Some expanders, such as the quad helix and the Schwartz, are best suited for slow palatal expansion in which the active period of expansion occurs over 4-6 months using 2 pounds of activation force [12]. Rapid expanders, such as the Haas and the Hyrax, allow for expansion at a much faster rate, completed in as little as 2 weeks, using 20 pounds of activation force [12]. As long as the expander used separates the midpalatal suture during treatment, there are no significant differences in treatment outcomes between different expander designs [9,11].

The Hyrax expander contains a jack screw in the center of the palate that is used to widen the appliance and put a transverse force on the posterior dentition to separate the palatine shelves [13]. A typical expansion protocol for a Hyrax is one turn per day for two weeks. Once the midpalatal suture has been sufficiently expanded to eliminate the transverse discrepancy, there is an enlargement of the local blood vessels leading to increases in inflammatory mediators that assist in bone formation thereby reconnecting the palatal shelves [14]. A study in dogs showed that once rapid expansion was completed, the midpalatal suture region was completely filled in by bone within 21 days [14]. In slow expansion, the rate of expansion itself is equal to the amount of time it takes for bone to be remodeled in the region. As a result, only 1-3 months of retention is required, but the overall treatment duration may be extended as a result of waiting to put on brackets and wires. With rapid expansion, the expansion duration is much faster than the remodeling process, so retention with archwires or a retainer is needed for at least 4-6 months after the active expansion period is over [12].

The sutural widening that occurs with maxillary expansion is pyramidal in shape [15]. This means that there is more expansion in the anterior than the posterior, and more inferiorly than superiorly. Essentially, the more skeletal resistance there is, the less an appliance can expand the arch. Because expansion is not entirely parallel, different teeth along the arch may be affected differently. As a result, the arch shape created with expansion may not necessarily be ideal and some teeth may be over-expanded or even in buccal crossbite compared to others. Orthodontic treatment following rapid palatal expansion is typically required to coordinate the arches and close the midline diastema. When expansion is completed in the mixed dentition, this pyramidal treatment effect may dramatically benefit the erupting canines, as they will have the greatest amount of space created for them in the transverse plane along the adjacent portion of the midpalatal suture and also anteriorly by consolidating the spacing created at the midline. This is very important because maxillary canines are the second most commonly impacted tooth after third molars, with 0.27-2.4% of people in the general population affected and 23.5% of patients who present to an orthodontic office for treatment [16,17]. After rapid palatal expansion, 70% of patients with a displaced and likely impacted cuspid saw an improvement in the eruption angulation and 40% had normal eruption of the tooth into the arch [17]. The canines are an excellent example of how expansion may influence the follicle orientation and success in permanent dental eruption.

### *Normal Dental Eruption*

The ability of the permanent canines to erupt into a normal occlusion following expansion treatment illustrates how perhaps some aspect of expansion, whether it is increasing the space available, changing the orientation of the deciduous teeth, or increasing the width of the skeletal base, may influence the eruption angulation of the permanent dental follicles. While this process may be susceptible to external influence, there is a large degree of genetic control. The orientation at which the teeth erupt is known to be controlled by a number of biologic factors and molecular signaling pathways [18]. As the developing tooth moves from the base of the crypt to the gingiva throughout the pre-eruptive stage of eruption, there is a polarization of signals from the dental follicle that promote osteoclasts to break down bone and form an eruption pathway coronally while osteoblasts build up bone apically. The elongation of the root has been found to accelerate, but not direct movement [19]. The dental follicle, a soft tissue sac encapsulating the developing tooth, is the single most important factor for dental eruption. Although the eruption process itself does not begin until dental calcification begins (between birth and 3 years for all permanent teeth [20]), most of the process is completely automated by the follicle and not the enamel organ. In fact, the follicle can erupt without any enamel, dentin, or root development inside [18]. A study by Cahill and Marks in dogs demonstrated that when the tooth bud was replaced by a metal substitute, the follicle still oriented itself and erupted normally [21]. In cases where there is complete root agenesis, the dental follicle also erupts normally [19].

Although the polarization of the follicle orients the erupting tooth towards the gingiva, there are several factors that may affect how this polarization occurs. Unlike orthodontic tooth movement, where the osteoblastogenesis and osteoclastogenesis work together across opposite sides of the tooth surface in a ‘coupled’ mechanism of action, these processes are ‘uncoupled’ in dental eruption. This means that the osteoblast activity at the follicle apex and the osteoclasts that breakdown alveolar bone above the follicle crown occur independently of one another. It is critical that there is a greater amount of bone formed in this stage than broken down. This allows the alveolus to grow in addition to the tooth erupting. How or whether there is any communication between the coronal and apical ends of the follicle is unclear, but this signal separation may make the erupting tooth more susceptible to disturbances in eruption or changes to the eruption vector. Once the tooth erupts, the dental follicle tissue forms the periodontal ligament, and it becomes capable of coupled communication during orthodontically influenced osseous remodeling [18].

The dental follicle is the only structure capable of stimulating alveolar bone growth [22]. The alveolus grows and expands around the developing tooth until it erupts and is generally maintained so long as the periodontium remains healthy and the tooth is not lost. In addition to the follicle signaling alveolar growth, the underlying alveolar and basal bone modeling may also have a limited capability to direct the tooth movement itself. Increases in maxillary basal bone width, maxillary cross-arch alveolar process width, and mandibular cross-arch alveolar process width during growth are closely associated with transverse molar movements and uprighting during growth [23]. Because the maxillary molar erupts buccally and uprights palatally, one would expect the intermolar distance to decrease as teeth continue to erupt. What is observed, however, is that as the basal and alveolar bone continues to widen, the roots follow this bony development and the archwidth continues to increase as the teeth upright [23]. When the maxilla is expanded in orthodontic treatment, these growth changes that increase the arch width and influence the posterior occlusal development are enhanced, and the underlying tooth buds are directly repositioned laterally as they erupt [5]. By translating the tooth buds to create a much larger arch width and creating more space for the maxillary molars to upright, early expansion promotes a favorable occlusion.

### *Environmental Influences on Dental Eruption*

There may be a number of external stimuli that have the ability to influence the eruption vector of the permanent teeth. Ectopic positioning of the primary teeth or the presence of a supernumerary tooth or odontoma, for example, may divert the course of eruption of the permanent teeth [16]. Normal maxillary canine eruption is heavily dependent on the morphology and position of both the adjacent permanent lateral incisors and first premolars [16,24]. The canine erupts mesially until its crown reaches the distal aspect of the lateral incisor root, at which point the crown distalizes to form a more upright eruption path [25]. When the lateral incisor is undersized or missing, there is a higher prevalence of canine impaction or ectopic eruption [16,26,27]. When there is a palatally displaced canine and more space is created for the erupting tooth through phase I orthodontic treatment, the permanent canine is significantly more likely to erupt normally than in patients who had no treatment. This occurs despite having no direct intervention through exposure and bonding of the impacted cuspid, as the phase I treatment generally only involves extracting the primary cuspid [24,28], extracting the primary cuspid and first molar [24], or extracting the primary cuspid in conjunction with and rapid palatal expansion and headgear [29,30]. There are also changes in the eruption vector of permanent teeth when deciduous teeth are used as anchors for tooth-borne expanders. In these patients, there is spontaneous uprighting of the erupted or erupting permanent first molars that were not directly involved with the appliance, as well as alleviation of dental compensations and anterior crowding [31–33]. This evidence suggests that in addition to the signaling cascade under genetic control that plays a large role in directing permanent tooth eruption, there may also be a substantial component that is influenced by the surrounding teeth and structures, which is susceptible to changes by orthodontic tooth movement.

### *Treatment Timing*

Timing of treatment is an important consideration for patients in need of maxillary expansion, as the mid-palatal suture continues to fuse with age. By the time a patient reaches the end of their adolescent growth spurt, the midpalatal suture is visibly radiopaque in CBCT scans [34]. This radiopacity corresponds to increased density caused by osseous bridging across the suture line that continues until the cessation of growth in late adolescence. As expansion aims to separate and widen the midpalatal suture, it is most successful prior to fusion or early on when there is only minor initial bridging. Otherwise, extensive microfracturing or even surgery is required to have a skeletal effect [5]. Patients late in adolescence whose midpalatal sutures have already begun to fuse, will have less of an orthopedic effect and more unfavorable dental side effects with expansion treatment [35,36]. Negative side effects associated with expansion in the permanent dentition [37] include dental tipping [38], alveolar bone bending [39], alveolar bone loss [40,41], gingival recession [42], root resorption [43,44], and white spot lesions [45]. In patients who have developed ‘superior convergent’ dental compensations, expansion in general may further tip the teeth in an unfavorable direction, especially if the patient is older and therefore more likely to experience dental side effects. Thus, maxillary expansion in patients with deciduous or mixed dentition may minimize dental side effects and maximize skeletal changes [10,31,37,46,47].

Treatment timing for mandibular dental expansion is also critical, as expansion in the mixed dentition may be more stable than in the permanent dentition [5]. Mandibular expansion may minimally increase the intercanine distance less than 2mm [48], however it is prone to relapse and greatly benefits from a bonded retainer. When expansion is done in the mixed or early mixed dentition, however, the intercanine dimension is greater from the onset of eruption. Studies of patients who have had mandibular expansion that increased the intercanine dimension in the mixed dentition demonstrate mixed results about whether the results were stable long term. While some studies show excellent stability [49], others show mild to moderate amounts of relapse [48,50]. A comparison of the mandibular intercanine width between patients who had maxillary expansion in the early mixed dentition and those who had no expansion show stable increases in the intercanine width in patients treated with maxillary expanders [31,51]. Expansion in the early mixed dentition may produce stable increases in the mandibular intercanine width, although mandibular overexpansion may still be prone to mild to moderate relapse.

Despite concerns about asymmetric growth and development, there is also evidence that transverse dental discrepancies observed before the age of six may be able to self-correct. In fact, in patients younger than 6 with unilateral posterior crossbites, 20% had spontaneous correction in the permanent dentition [4]. This same study observed that 8% of patients who had no crossbites in the primary dentition developed them later in the permanent dentition. This fluctuant malocclusion illustrates how timing-dependent orthodontic treatment is. Malocclusion in the primary dentition is not necessarily indicative of a skeletal deficiency and may not be concerning for growth if observed too long before the adolescent growth spurt. The mixed dentition gives a much clearer picture of whether any early dental or skeletal treatment is needed and is a much more opportune time to begin treatment.

***Growth Analysis***

Expansion has clear therapeutic benefits for patients with maxillary constriction when treatment is initiated prior to the cessation of growth. As many of these patients are being treated just prior to their major adolescent growth spurt, any analysis of the dentition throughout expansion must consider the growth changes that overlap with the timing of treatment. Around the same time that expansion treatment is indicated, the condyle grows upward and backward (although this is subject to individual variation and sometimes grows forward). This typically causes the anterior portion of the mandible mandible to move downwards and forward during the adolescent growth spurt [5, 52]. The maxilla is also modeled in early adolescence, as the anterior nasal spine moves downwards and forwards[5]. Geometric morphometric analysis and principal component analysis are imperative to accurately measure the eruption vector of the permanent teeth while controlling for these growth and positional changes.

Geometric morphometric analysis is used to determine shape differences between two groups while controlling for scalar differences. This type of analysis can easily control for growth and development occurring simultaneous to any treatment intervention by generating a set of procrustes coordinates in which all landmarks are adjusted to be relative to the same grid. Previous studies have used geometric morphometric analysis to demonstrate that the shape and width of the maxillary and mandibular dental arches are most closely related to each other [53], and also that the palatal width and shape (and by extension the maxillary arch width) is strongly related to the mandibular plane angle [54]. This type of analysis is critical for demonstrating how orthodontic treatment affects occlusion across all three planes of space, while controlling for growth and positional changes.

Principal component analysis takes the information gleaned from the geometric morphometric analysis one step further. This statistical analysis determines how much of the variance in a sample is explained by a set of linearly uncorrelated variables. These variables can then be removed in order to transform the data into a set of linearly correlated variables. In a study on dental eruption, for example, principal component analysis can distinguish between growth changes and dental treatment effects. The growth or positional changes can then be separated from the data set to determine the actual dental changes that occurred throughout treatment. For patients treated with rapid palatal expansion around the same time as their adolescent growth spurt, understanding the differences between growth and orthodontic treatment effects is critical for meaningful results.

***Objective***

The purpose of our study is to evaluate whether expansion in the early mixed dentition when all primary canines and molars are present will change the eruption vector of the permanent canines and premolars. There is evidence that environmental influences can moderate the orientation at which permanent teeth erupt. Expansion in the primary and early mixed dentition is associated with more upright maxillary molars and a wider transverse arch width [23, 32]. Extracting deciduous canines or using headgear to create space for erupting permanent canines is associated with lower prevalence of impactions and more ideal eruption angulation[24,28,29,30]. If expansion in the early mixed dentition does change the eruption vector of the permanent canines and premolars, then the process of eruption may be subject to environmental influences more than we know. The erupting dental follicles may be able to create more lateral alveolar bone as they erupt across a greater transverse dimension. This widened arch may be more structurally sound to support more space for the tongue and the nasal airways. The amount of treatment needed in phase II may be reduced if the teeth are able to upright into an ideal angulation on their own, which may reduce the side effects associated with treatment. There may be many benefits to expansion in the early mixed dentition if the permanent tooth buds are indirectly affected. Our study seeks to determine whether the eruption vector of permanent canines and premolars are altered when expansion is completed in the early mixed dentition.

## Materials and Methods

This retrospective clinical study evaluates whether there is a difference in the eruption vectors of permanent canines and premolars in children who have had phase I expansion compared to children who have had no treatment. This study was conducted at UCSF in conjunction with a private practice in Mill Valley, CA. Ethical approval was obtained through the UCSF IRB board (IRB #10-00564) and all CBCT data was anonymized prior to analysis.

The patients included in this study (N=42) were 45.2% female (N=19) and 54.8% male (N=23). The participants were 75.6% Caucasian (N=31), 12.2% Hispanic (N=5), 4.8% Asian (N=2), and 9.8% other (N=4). CBCTs were taken on an iCAT FLX machine (Henry Schein Dental, Melville, NY) at 120 kVp, 5 mA, 16x13 cm, and 0.3mm voxel size at two timepoints. The first time point was between the ages of 7-11 years (mean age = 8.7 years) in the mixed dentition, all primary canines and primary molars were present. This was part of a routine diagnostic evaluation, which was completed for all patients, regardless of whether they ended up starting treatment. The second time point (mean age = 12.0 years) was before comprehensive or phase II orthodontic treatment once the patient was in the permanent dentition.

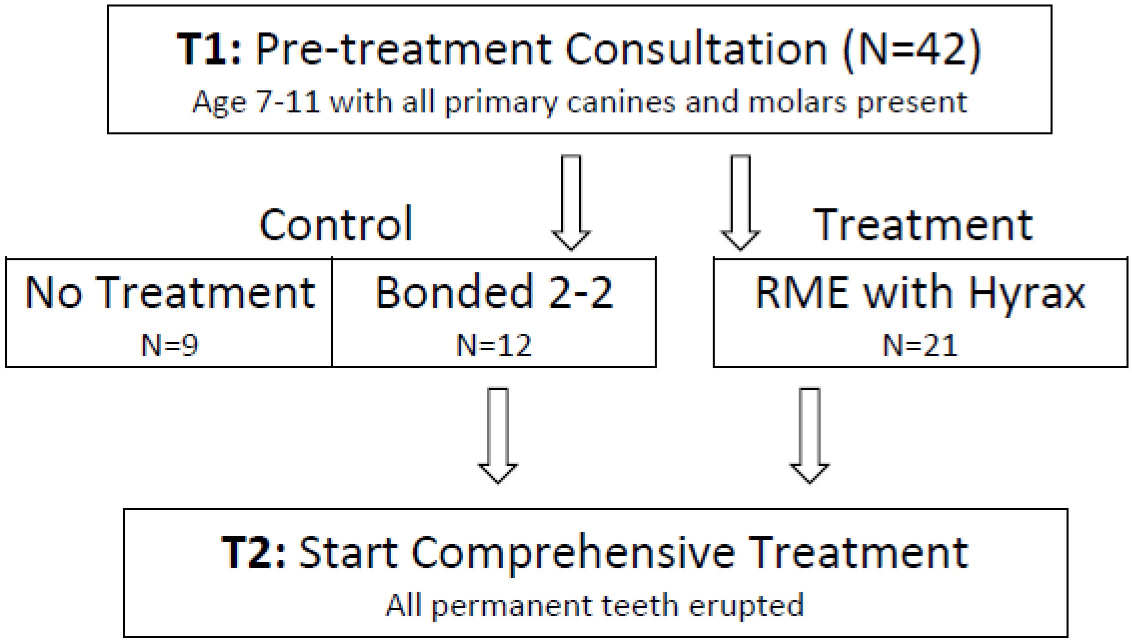
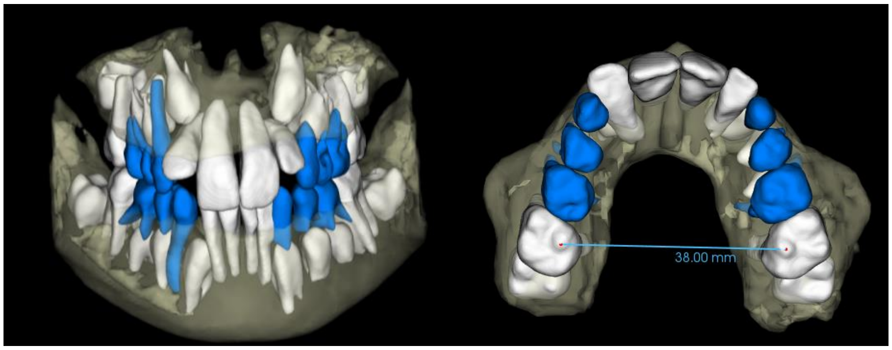
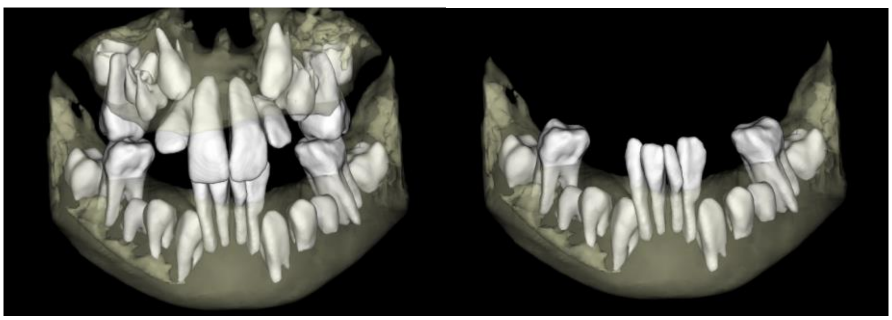



***Figure 1. Workflow***

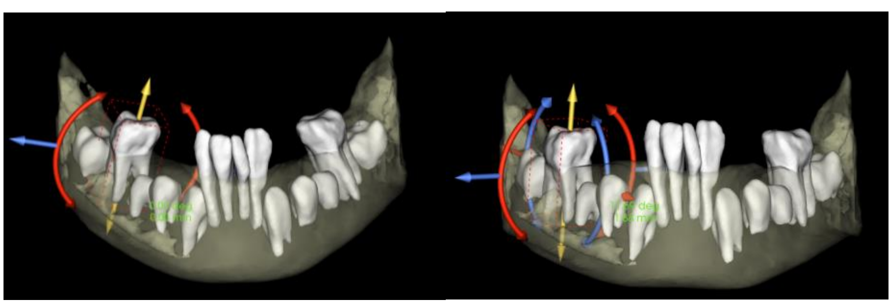
The treated group (N=21) had maxillary expansion using a Hyrax appliance bonded on the upper permanent first molars and mandibular expansion using an active lower lingual arch bonded on the lower permanent first molars. The expansion protocol was based on the individual patient's need, which was determined through a treatment rehearsal using Anatomodel software (Anatomage, San Jose, CA). This treatment rehearsal involved uprighting the lower permanent first molars over the alveolus and then expanding maxilla until the upper permanent first molar transverse position fit ideally with the new lower molar position. No participants in the treatment group had any additional orthodontic or orthopedic appliances in conjunction with expansion therapy. The control group of this study (N=21) had a CBCT taken as part of their diagnostic records while in the mixed dentition but did not receive any maxillary expansion. 12 of the 21 control patients (57.2%) had no treatment and 9 of the control patients (42.8%) were treated with limited fixed appliances on the incisors (with nothing bonded on the premolars or canines involved in this study).



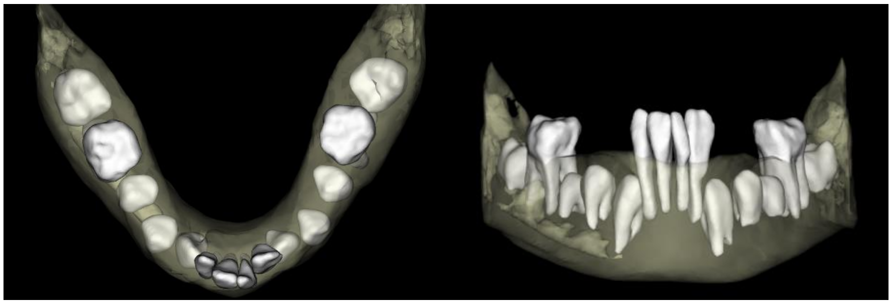

1. Measure the pre-treatment maxillary arch width



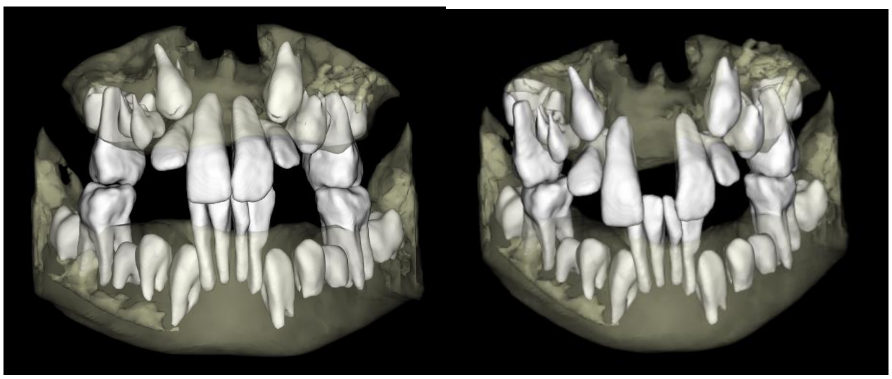
2. Remove all deciduous teeth, Isolate the lower arch



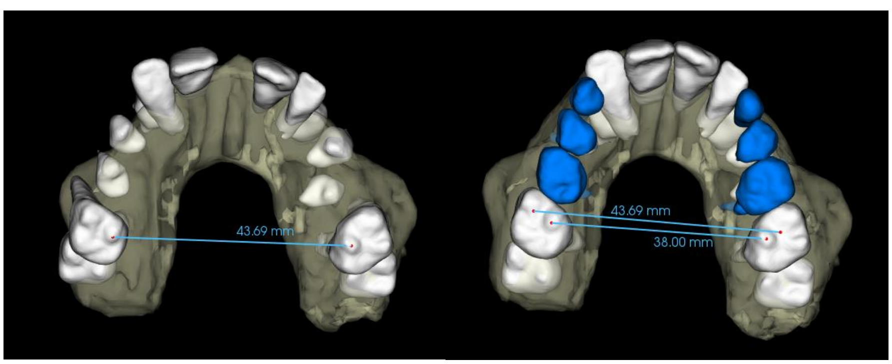
3. Upright lower first molars in the center of the alveolus



(Uprighted Lower molars centered in the alveolus)



4. Expand the upper arch to occlude with the upright lower molars




5. Measure the archwidth difference (mm) from the initial to the final

6. Mm needed x 4 turns/mm = Prescribed number of turns

***Figure 2. Treatment Rehearsal***

Children with severe malocclusions, including irregular sagittal or vertical skeletal growth, anterior or posterior crossbites, or severe spacing or crowding in the mandibular arch (Mean mandibular crowding = 0.8mm, range -2 to 4mm) were excluded. No patients had a history of orthodontic treatment prior to this study. The average overbite at start was 57.4% overlap.

CBCT scans were landmarked by one orthodontic resident using Invivo 5 software (Anatomage, San Jose, CA). Intra-rater reliability testing was completed across 5 CBCT records to ensure reproducibility in placing landmarks. Dental and skeletal landmarks were placed to measure the long axes of the canines and premolars relative to the cranial base. Initial and final scans were superimposed on the cranial base using manual landmarks and a voxel-based registration. 3D data points were then transferred to MorphoJ Geometric Morphometric software (Klingenberg lab, Manchester, UK) to standardize the superimpositions across all subjects in this study, generating a list of procrustes coordinates that control for scalar differences between images.

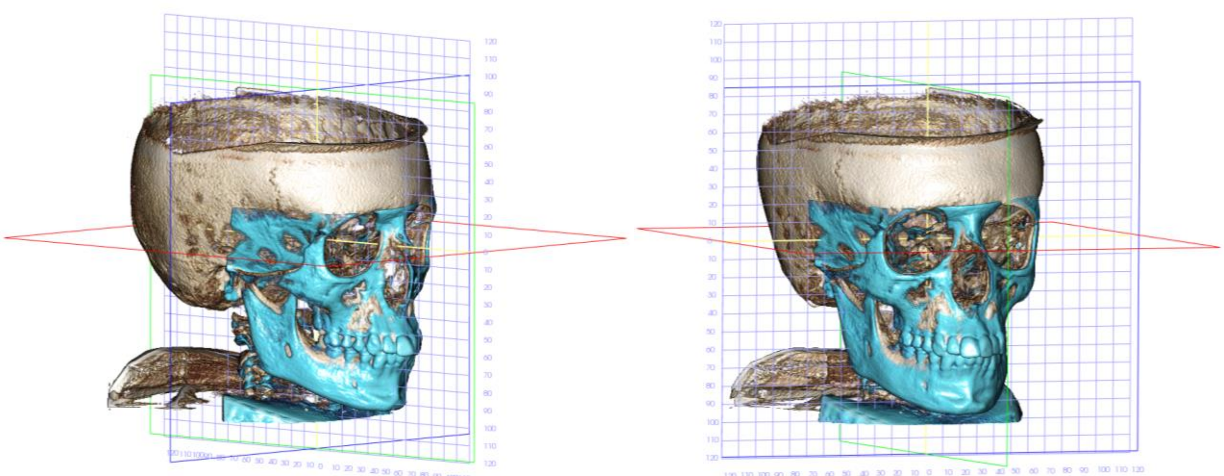
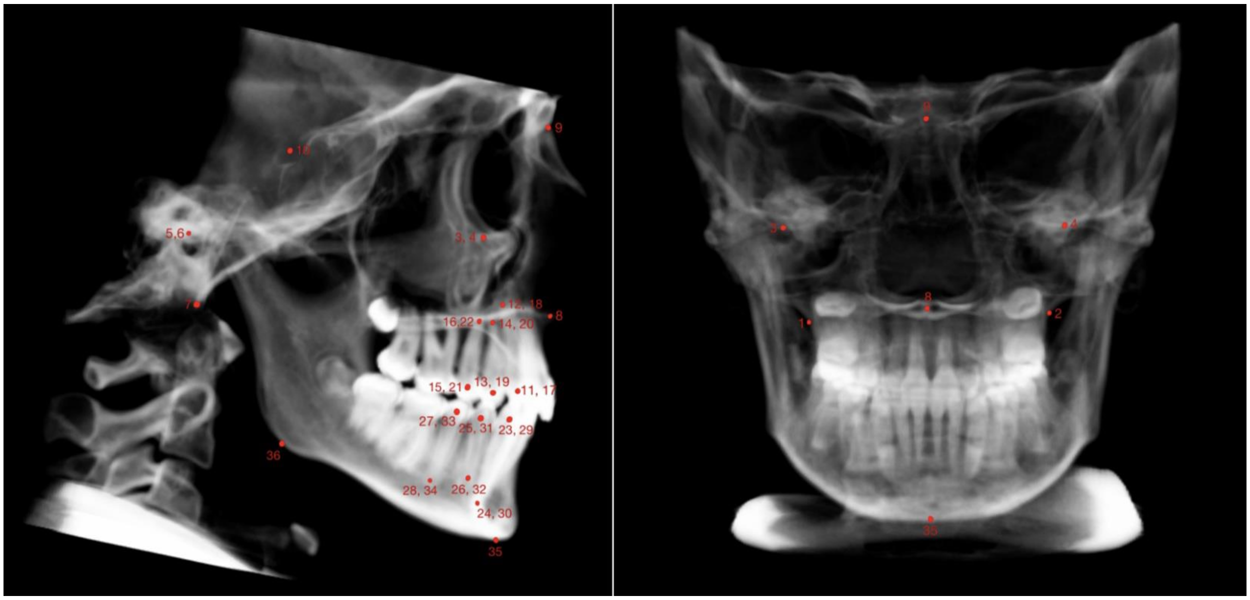


***Figure 3. Superimpositions in Invivo 5***




| | | |
|---|---|---|
| 1 | J Point (Right) | The deepest point in the concavity of the posterior maxilla as viewed in a coronal slice through the molars |
| 2 | J Point (Left) | |
| 3 | Orbitale (Right) | The lowest point on the infraorbital margin of each orbit |
| 4 | Orbitale (Left) | |
| 5 | Porion (Right) | The highest point on the external auditory meatus |
| 6 | Porion (Left) | |
| 7 | Basion | The midpoint of the anterior rim of the foramen magnum on the occipital bone |
| 8 | Anterior Nasal Spine | The most anterior point of the nasal floor |
| 9 | Nasion | The intersection of the nasofrontal and internasal sutures |
| 10 | Sella | The lateral and horizontal midpoint of the sella turcica |
| 11 | Upper Right Canine Crown | The center of the widest portion of the canine crown both buccolingually and mesiodistally |
| 12 | Upper Right Canine Apex | The center of the root apex, both buccolingually and mesiodistally |
| 13 | Upper Right First Premolar Crown | The center of the widest portion of the premolar crown both buccolingually and mesiodistally |

| | | |
|---|---|---|
| 14 | Upper Right First Premolar Apex | The center of the root apex, both buccolingually and mesiodistally. If there is more than one root, the center is estimated as the midpoint between the two roots. |
| 15 | Upper Right Second Premolar Crown | The center of the widest portion of the premolar crown both buccolingually and mesiodistally. |
| 16 | Upper Right Second Premolar Apex | The center of the root apex, both buccolingually and mesiodistally. If there is more than one root, the center is estimated as the midpoint between the two roots. |
| 17 | Upper Left Canine Crown | The center of the widest portion of the canine crown both buccolingually and mesiodistally |
| 18 | Upper Left Canine Apex | The center of the root apex, both buccolingually and mesiodistally |
| 19 | Upper Left First Premolar Crown | The center of the widest portion of the premolar crown both buccolingually and mesiodistally |
| 20 | Upper Left First Premolar Apex | The center of the root apex, both buccolingually and mesiodistally. If there is more than one root, the center is estimated as the midpoint between the two roots. |
| 21 | Upper Left Second Premolar Crown | The center of the widest portion of the premolar crown both buccolingually and mesiodistally |
| 22 | Upper Left Second Premolar Apex | The center of the root apex, both buccolingually and mesiodistally. If there is more than one root, the center is estimated as the midpoint between the two roots. |
| 23 | Lower Right Canine Crown | The center of the widest portion of the canine crown both buccolingually and mesiodistally |
| 24 | Lower Right Canine Apex | The center of the root apex, both buccolingually and mesiodistally |
| 25 | Lower Right First Premolar Crown | The center of the widest portion of the premolar crown both buccolingually and mesiodistally |
| 26 | Lower Right First Premolar Apex | The center of the root apex, both buccolingually and mesiodistally |
| 27 | Lower Right Second Premolar Crown | The center of the widest portion of the premolar crown both buccolingually and mesiodistally |
| 28 | Lower Right Second Premolar Apex | The center of the root apex, both buccolingually and mesiodistally |
| 29 | Lower Left Canine Crown | The center of the widest portion of the canine crown both buccolingually and mesiodistally |
| 30 | Lower Left Canine Apex | The center of the root apex, both buccolingually and mesiodistally |

| | | |
|---|---|---|
| 31 | Lower Left First Premolar Crown | The center of the widest portion of the premolar crown both buccolingually and mesiodistally |
| 32 | Lower Left First Premolar Apex | The center of the root apex, both buccolingually and mesiodistally |
| 33 | Lower Left Second Premolar Crown | The center of the widest portion of the premolar crown both buccolingually and mesiodistally |
| 34 | Lower Left Second Premolar Apex | The center of the root apex, both buccolingually and mesiodistally |
| 35 | Menton | The lowest point of the symphysis of the mandible |
| 36 | Gonion | The estimated midpoint of the posterior angle of the mandible. |

*All landmarks were identified both on a 3D CBCT image and on 2D slices through all 3 planes of space

***Figure 4. Radiographic Landmarks***

Angular changes between the initial and final long axes were calculated using a modified Pythagorean Theorem. This equation superimposed the apices of each tooth at the initial and final timepoints to calculate the absolute change in angulation in 3D.

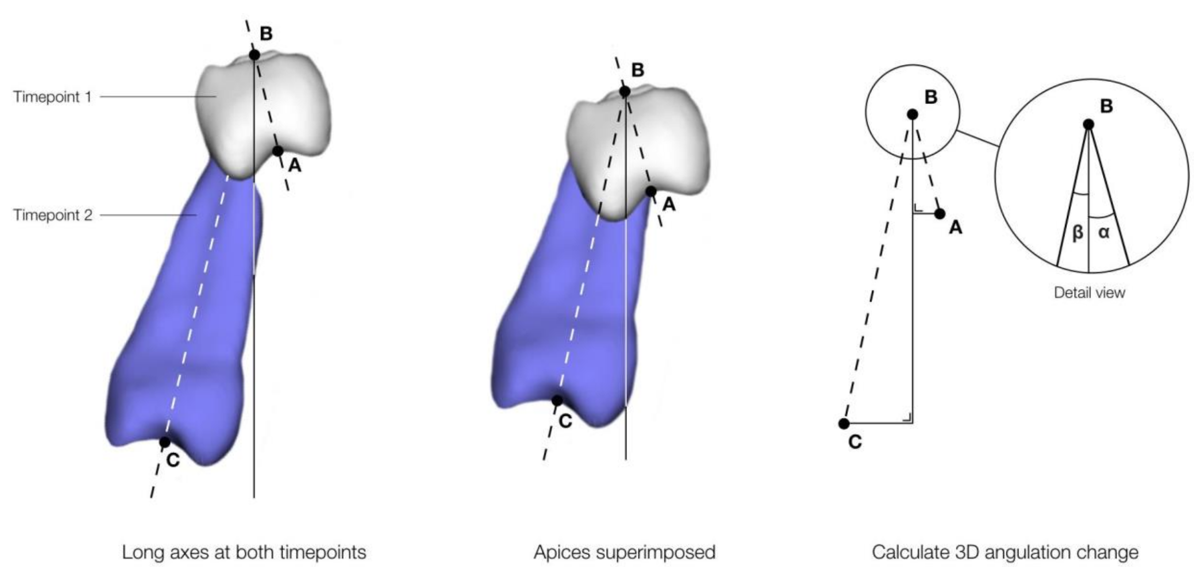


***Figure 5. Modified Pythagorean Theorem***
*2D representation of the 3D Modified Pythagorean Theorem used to calculate angulation differences between initial and final long axes of the permanent teeth*

Statistical analyses on the procrustes coordinates generated from the geometric morphometric analysis in MorphoJ software were completed with SPSS software version 21 (IBM, Armonk, NY). Paired t-tests were used to compare the angular change in degrees across the two timepoints between the control and treated groups. Principal component analysis was then completed using MorphoJ software.

## Results

Landmarks were converted into procrustes coordinates using MorphoJ software (Klingenberg lab, Manchester, UK) to control for scalar, translational, and rotational differences. Procrustes coordinates were graphed in MorphoJ and represented in 2D across 3 axes (see figure 6).

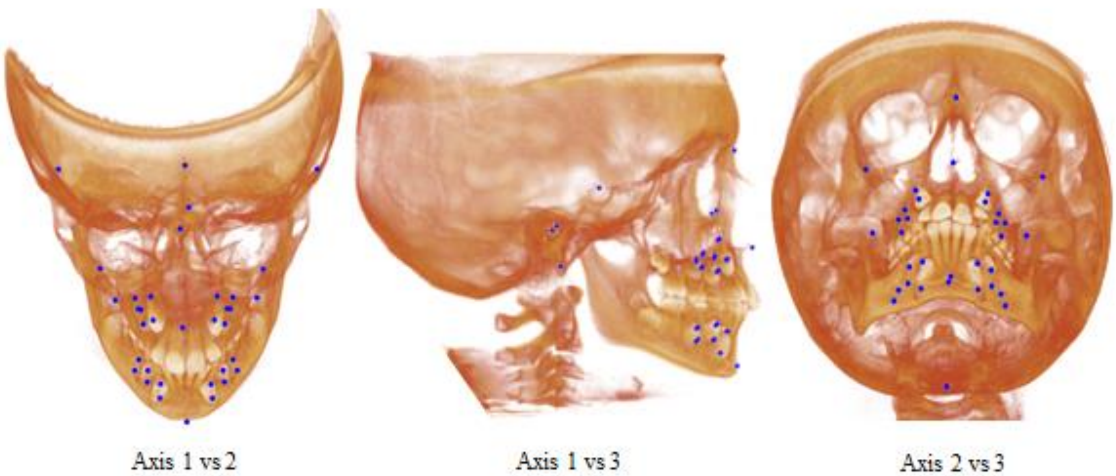



***Figure 6. MorphoJ landmarks on 3D CBCT image.***
*Axis 2 vs 3 was used in the figures of this study to describe the transverse position of the teeth*

Figure 6 compares the landmark graphs of principal component 1 to the 3D CBCT that it represents. This landmark graph illustrates the average positions of the dental and some of the skeletal landmarks used in this study. Figure 7 illustrates how the dental landmarks represent the long axis of the teeth when the landmarks indicating the center of the crown and center of the apex are connected.

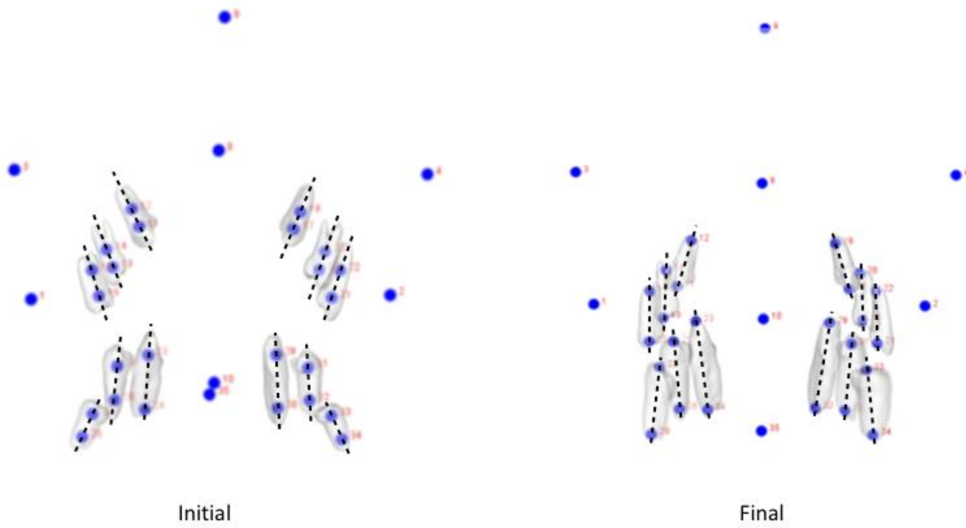
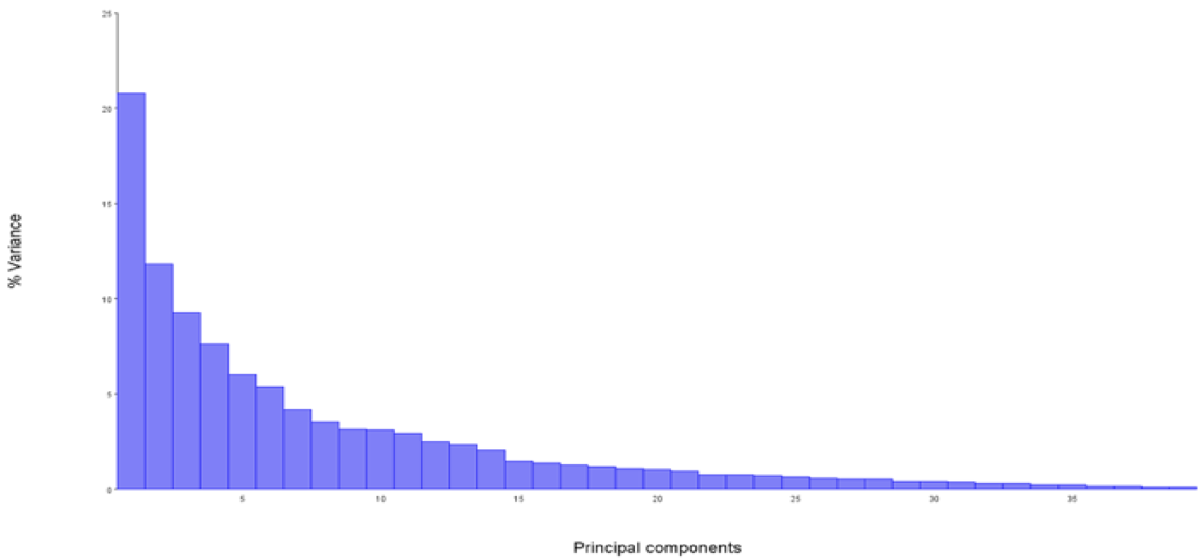



***Figure 7. MorphoJ dental landmarks: PC1, initial and final***
*The intraoral landmarks with model teeth overlaid demonstrate how the canines, first premolars, and second premolars are illustrated by the points on landmark graphs. Each tooth has a landmark at the center of the crown and at the center of the apex, forming its long axis when combined. These landmark graphs are axis 2 vs 3 of principal component 1 with a scale factor of 0 at the initial and final timepoints.*

The control group of this study was composed of patients who either had no treatment or had limited phase I treatment with no expansion and nothing bonded to the canines and premolars. There were no statistically significant differences in the eruption vector of canines and premolars for patients who had and did not have limited non-expansion phase I treatment (p=0.24). There were also no significant differences between the control group and treatment group at the initial timepoint (p=0.5976), although a comparison of the different principal components showed slight, non-significant differences between principal component 5 and principal component 7. Figure 8 demonstrates that 50% of the shape variance in the initial sample was explained by principal components 1 through 4. All principal components prior to PC14 were responsible for greater than 2% of the variance in the sample, and most of the variance observed following PC14 was noise.

None of the main 4 principal components showed any differences with the 14 relevant principal components. Figure 9 illustrates how principal component 5, responsible for 7% of shape variation at the initial timepoint, and principal component 7, responsible for 4% of variation, were the only two distinct principal components. The landmark graphs of these principal components illustrate that the shape differences between these groups are negligible. Although these principal components demonstrate some shape differences, they are not statistically significant, they are both only responsible for a small percentage of the variation in the sample, and the morphological differences observed are negligible.



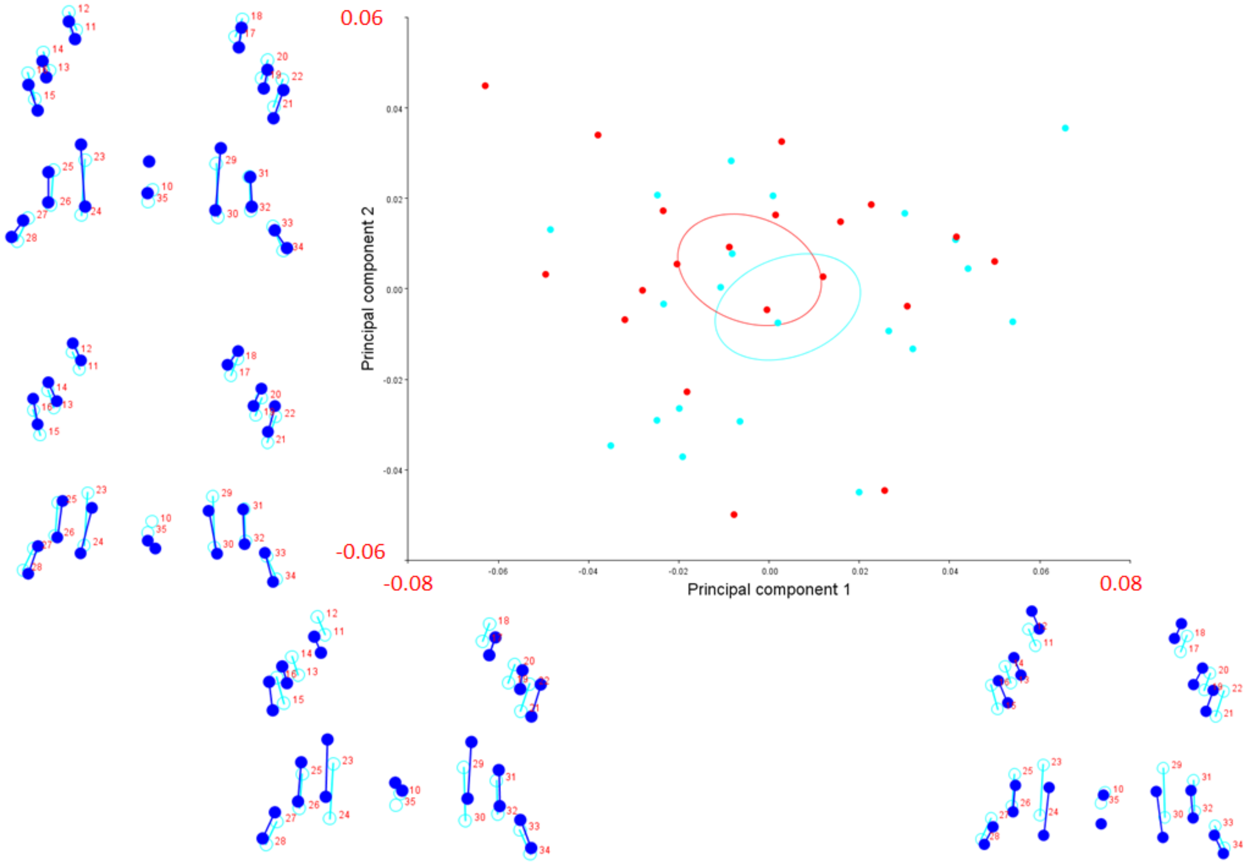



***Figure 8. Principal Component Analysis: Initial Timepoint, PC1 and PC2***
*A) PC1 explained 21% of variation in the shape of the skull and dental arch. PC2 explained 12% of variation. Principal components 1-14 were all responsible for greater than 2% of the variance in the sample.*
*B) As PC1 increased, the teeth moved from being more upright to mesially angulated. As PC2 increased, the teeth became more upright. There were no major differences observed between the control group (red) and treatment group (blue) for principal components 1 and 2 (p=0.5976).*

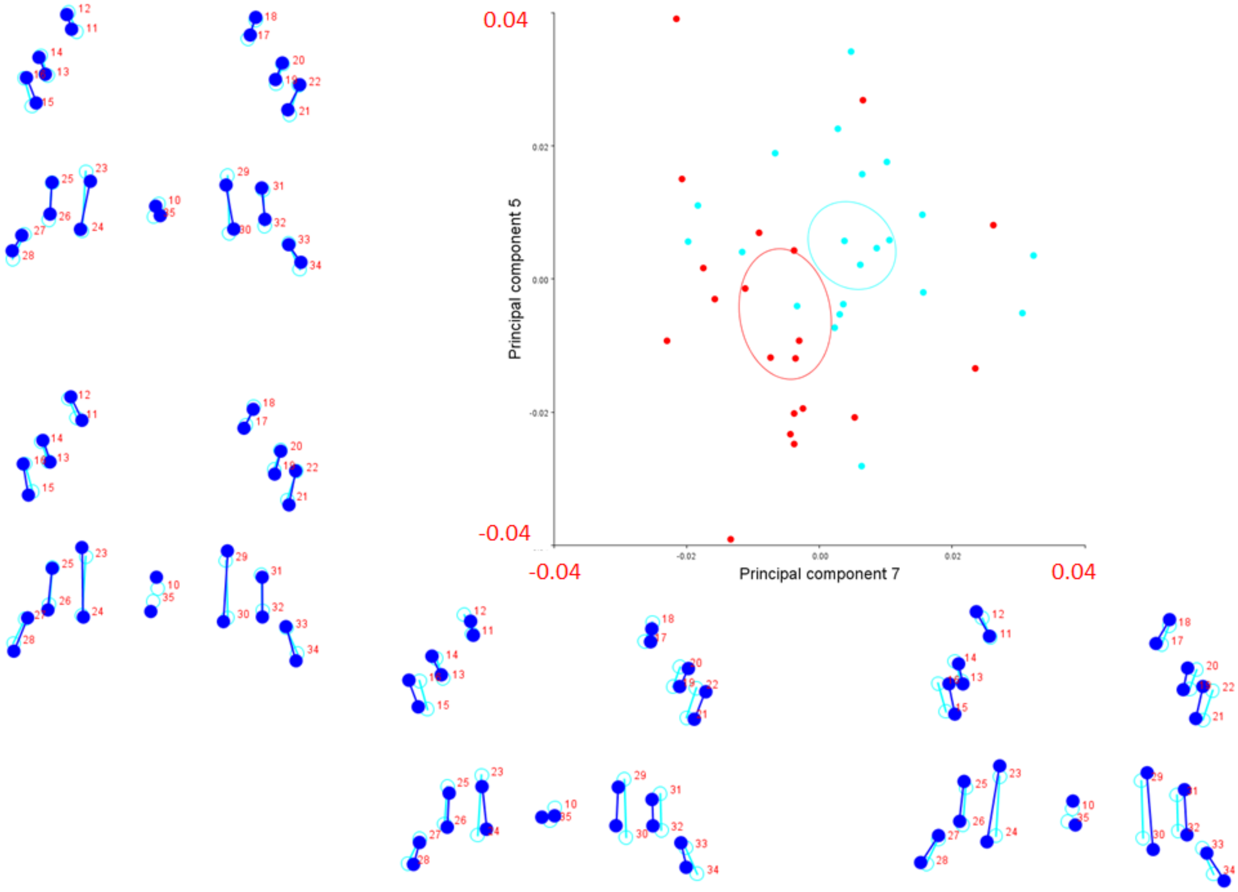



***Figure 9. Principal Component Analysis: Initial Timepoint, PC5 and PC7***
*PC5 explained 7% of the variation in the shape of the skull and dental arch. PC7 explained 4% of variation. These were the only two principal components at the initial timepoint that demonstrated minor shape differences, however these differences were not statistically significant (p=0.5976).*

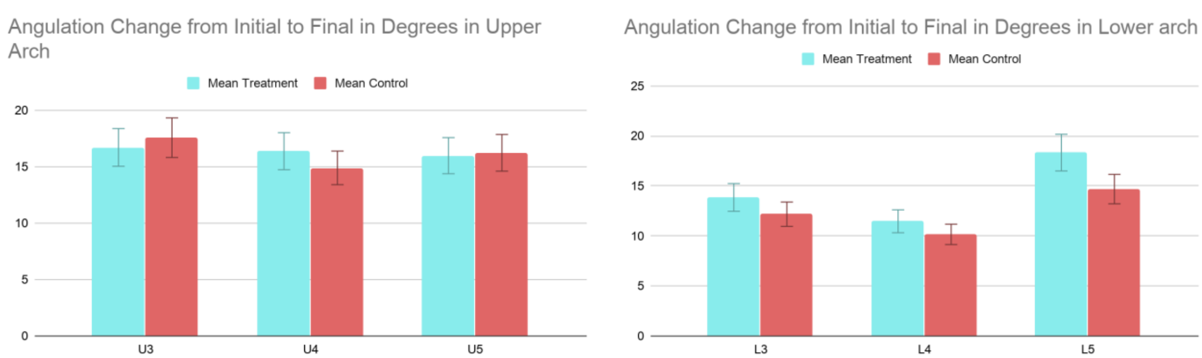



***Figure 10. Angulation changes between initial and final in degrees.***

*A) The upper canines demonstrated a greater angulation change in the control group than the treatment group (p=0.27). The upper first premolars showed the opposite, with a greater angulation change in the treatment group than the control group (p=0.15). There was no change in angulation for the upper second premolars (p=0.50). None of the differences observed in the upper arch were statistically significant (p>0.05).*
*B) The amount of uprighting on the lower arch was less in the control group than the treatment group for the lower canines (p=0.26), lower first premolars (p=0.23), and lower second premolars (p=0.06). None of the differences observed in the lower arch were statistically significant (p>0.05).*

Figure 10 shows the angular change between timepoints for each tooth in the control group and treatment group. These values were calculated using the procrustes coordinates in a modified Pythagorean theorem. None of these differences were statistically significant. The lower ach consistently had less angulation change in the control group than the treatment group. The upper first premolar also followed this trend. The upper canine showed the opposite pattern, with more of an angular change in the control group than in the treatment group. There were no differences observed between groups for the angulation of the upper second premolar. This evaluation demonstrated that there were angular changes between the initial and final time points, and that the amount of uprighting was different between the control group and treatment group for all of the teeth except for the upper second premolar. Although it identified a difference between the treatment groups, this analysis did not describe directionality. The principal component analysis was needed to accurately describe the positional differences that occurred. Figures 11 and 12 describe the shape differences observed between groups at the final time point. This principal component analysis included an additional regression for age and controlled for treatment time. Principal component 2, responsible for 14% of the shape variation in the sample, and principal component 8 demonstrated non-significant shape differences (p=0.0760). There were also slight differences observed without distinct separation of the means ellipses between principal component 2 and principal components 12, 13, 14, and 17. As PC2 increased, the roots on the upper teeth appeared more upright.

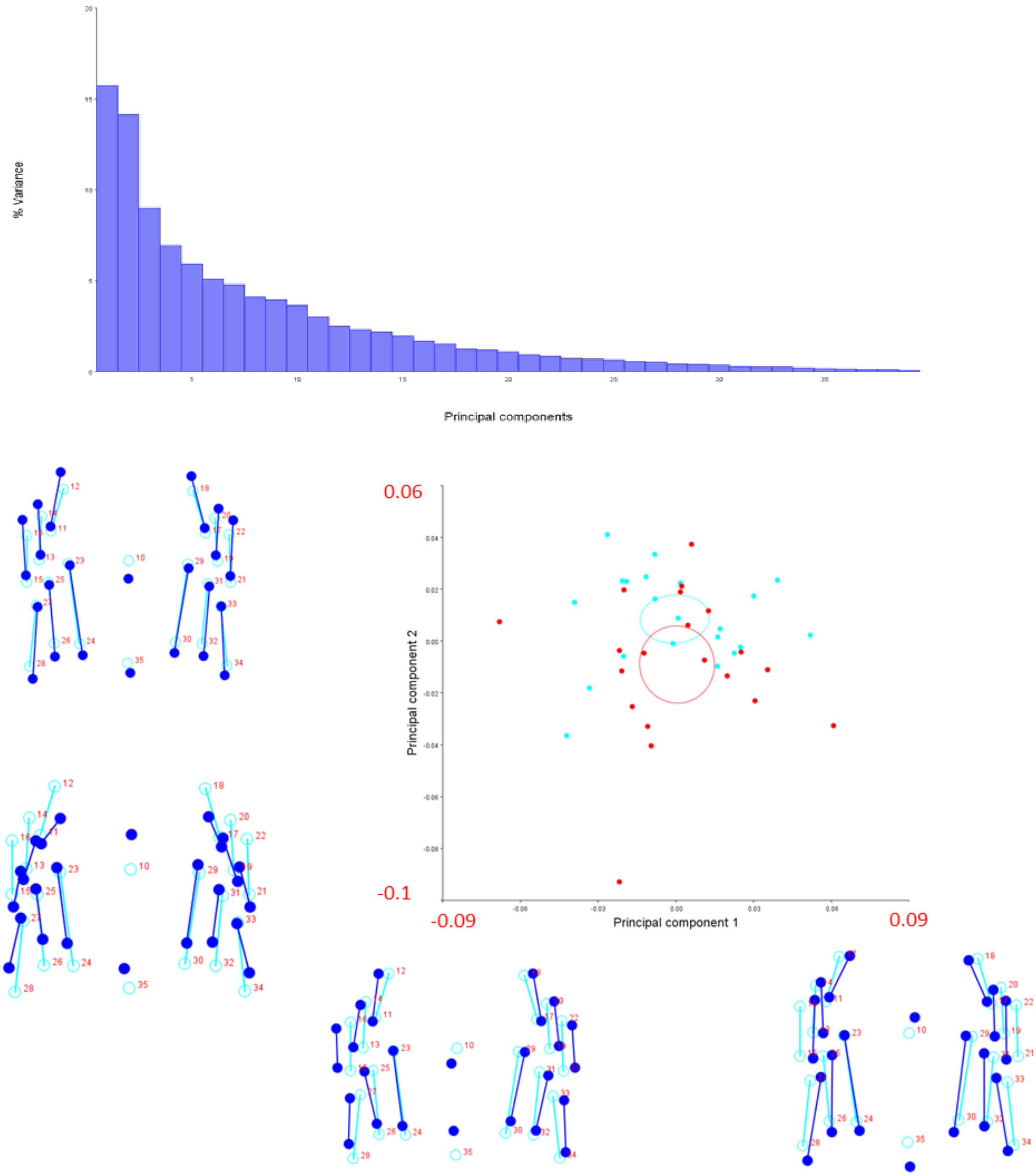



***Figure 11. Principal Component Analysis with age regression: Final Timepoint, PC1 and PC2***
*A) PC1 was responsible for 16% of variation in shape of the skull and dental arch. PC2 was responsible for 14% of variance.*

*B) There were no significant differences in shape between groups across PC1 and PC2. As principal component 1 increased, there was increased palatal root torque on the upper and lingual crown torque on the lower. As PC2 increased, the upper roots uprighted buccally and the lower roots elongated with their orientation roughly maintained.*

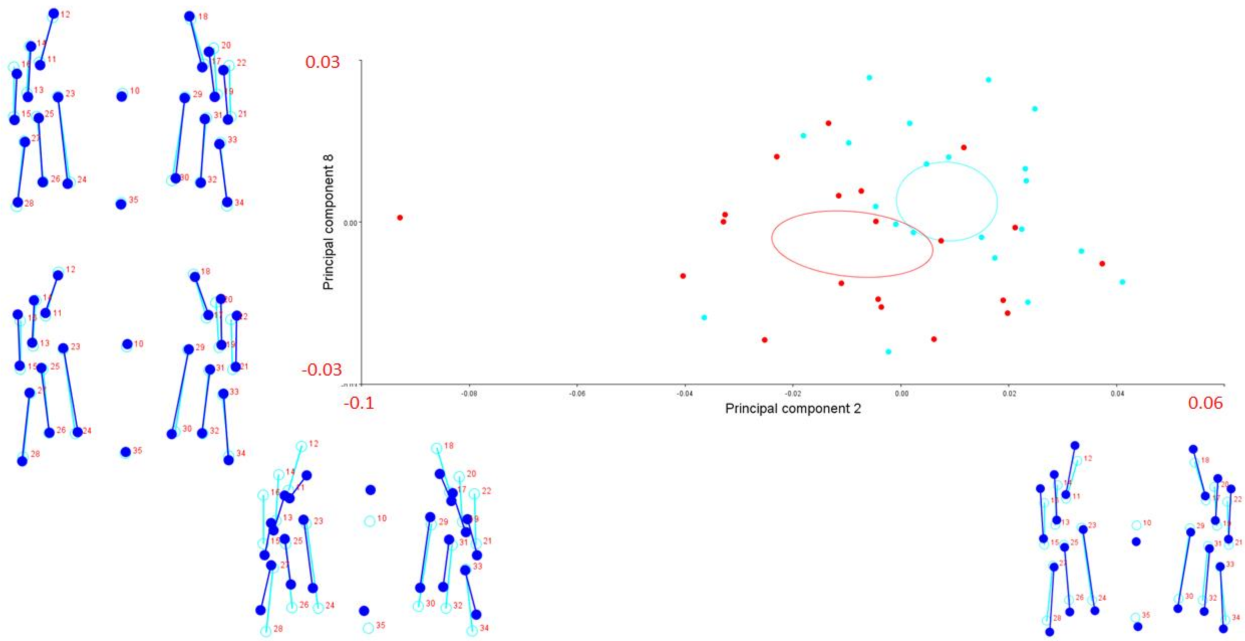



***Figure 12. Principal Component Analysis with age regression: Final Timepoint, PC2 and PC8***
*PC2 was responsible for 14% of shape variation. PC8 was responsible for 4% of shape variation. These principal components showed nearly significant separation between the control and treatment groups (p=0.0760). There were also mild differences observed without distinct separation of the means ellipses between principal component 2 and principal components 12, 13, 14, and 17.*

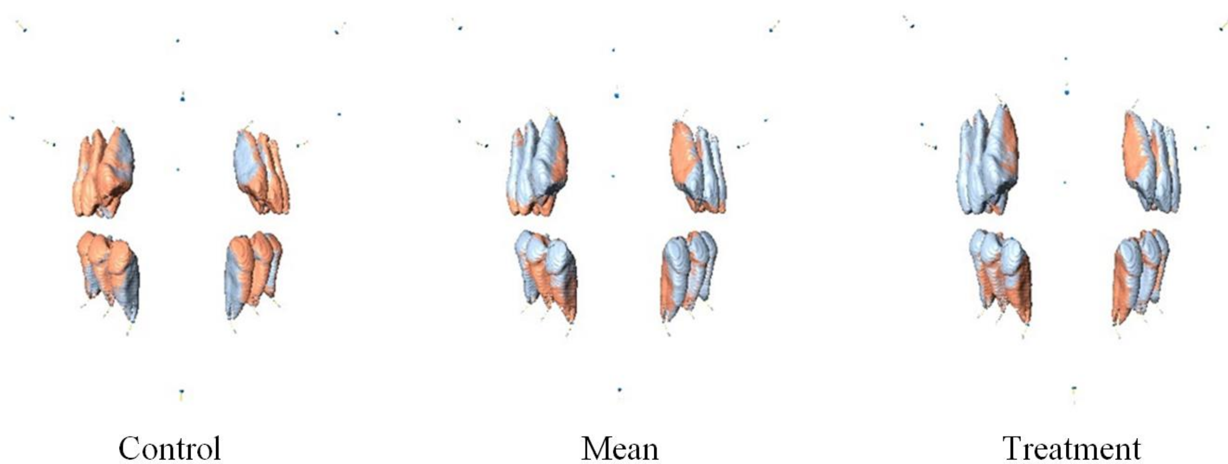



***Figure 13. Dental rendering of principal component 2***

*The red figure on the left represents the control group. It shows the average position of the canines and premolars according to principal component 2, which were buccally tipped. The blue figure on the right represents the treatment group. It shows that the average position of the teeth was more upright than in the control group figure. The center diagram shows the average angulation of the canines and premolars across all samples.*

When principal component 2 was stitched with a CBCT model, we were able to see how the angulation of the teeth differed between the treatment group and control group. Figure 13 shows three distinct images of the dental arch. The center image illustrates the average position of landmarks according to principal component 2. The teeth were slightly tipped buccally on both the upper and lower. The figure on the left in red represents the control group, while the figure on the right in blue represents the treatment group. The position of the teeth in the treatment group were more upright, whereas the position of the teeth in the control group were more tipped out. This principal component illustrates that the permanent teeth erupted in a slightly more upright orientation following phase I expansion.

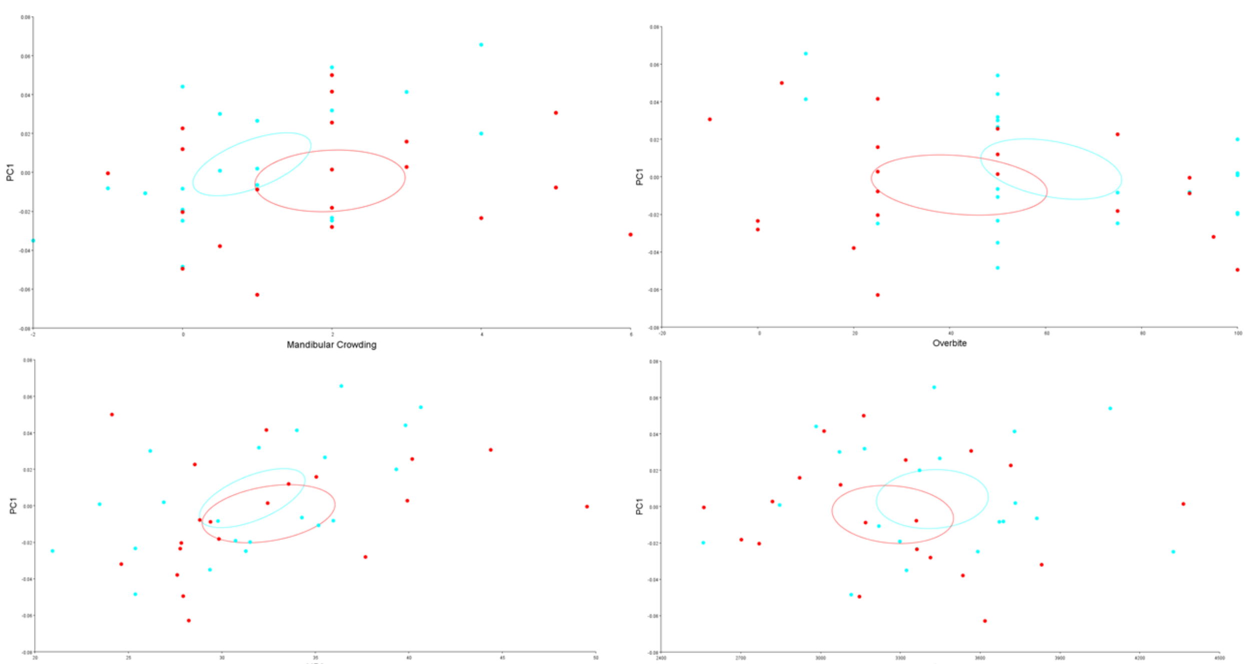


***Figure 14. Initial Covariation***
*The amount of mandibular crowding, overbite, mandibular plane angle (MP-SN), and age had no influence on shape differences in any principal component at the initial time point. This graph shows that there are no differences in principal component 1.*

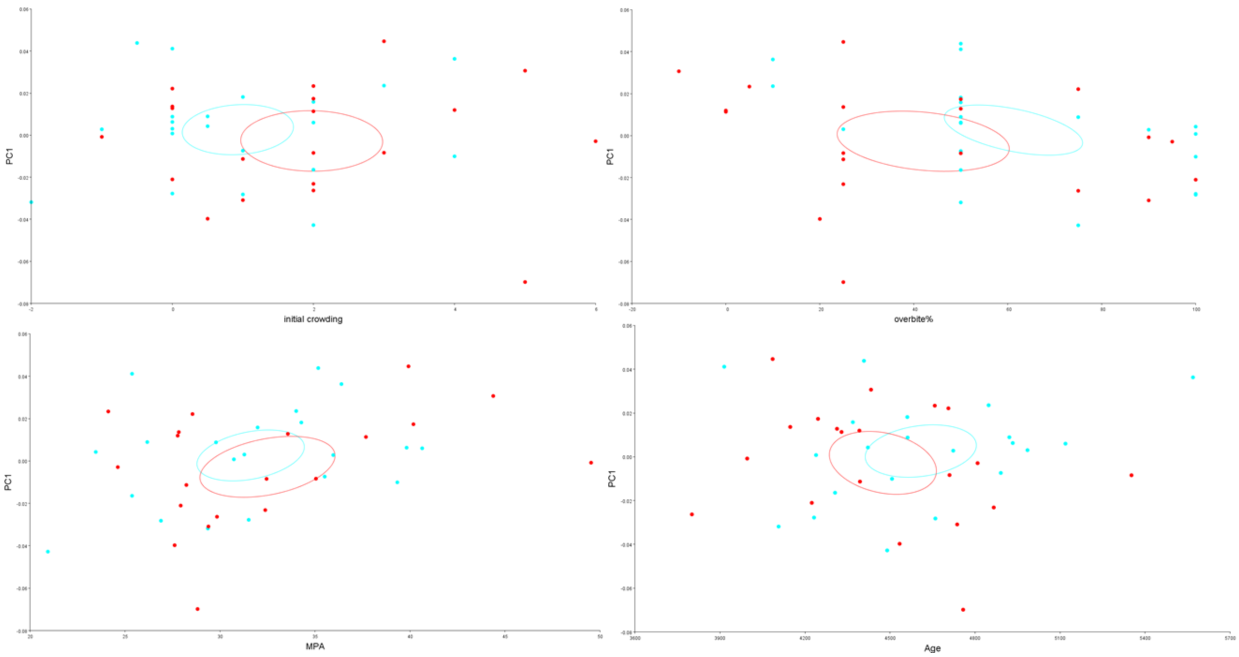



***Figure 15. Final Covariation***
*The amount of mandibular crowding, overbite, mandibular plane angle (MP-SN), and age had no influence on shape differences in any principal component at the final time point. This graph shows that there are no differences in principal component 1.*

Potential covariates were evaluated to determine if there may be other factors influencing shape differences between groups. The amount of initial crowding, overbite, mandibular plane angle, and age when starting treatment had no influence on shape differences between groups in any of the principal components at either time point. Figures 14 and 15 show the relationship between these covariates and principal component 1.

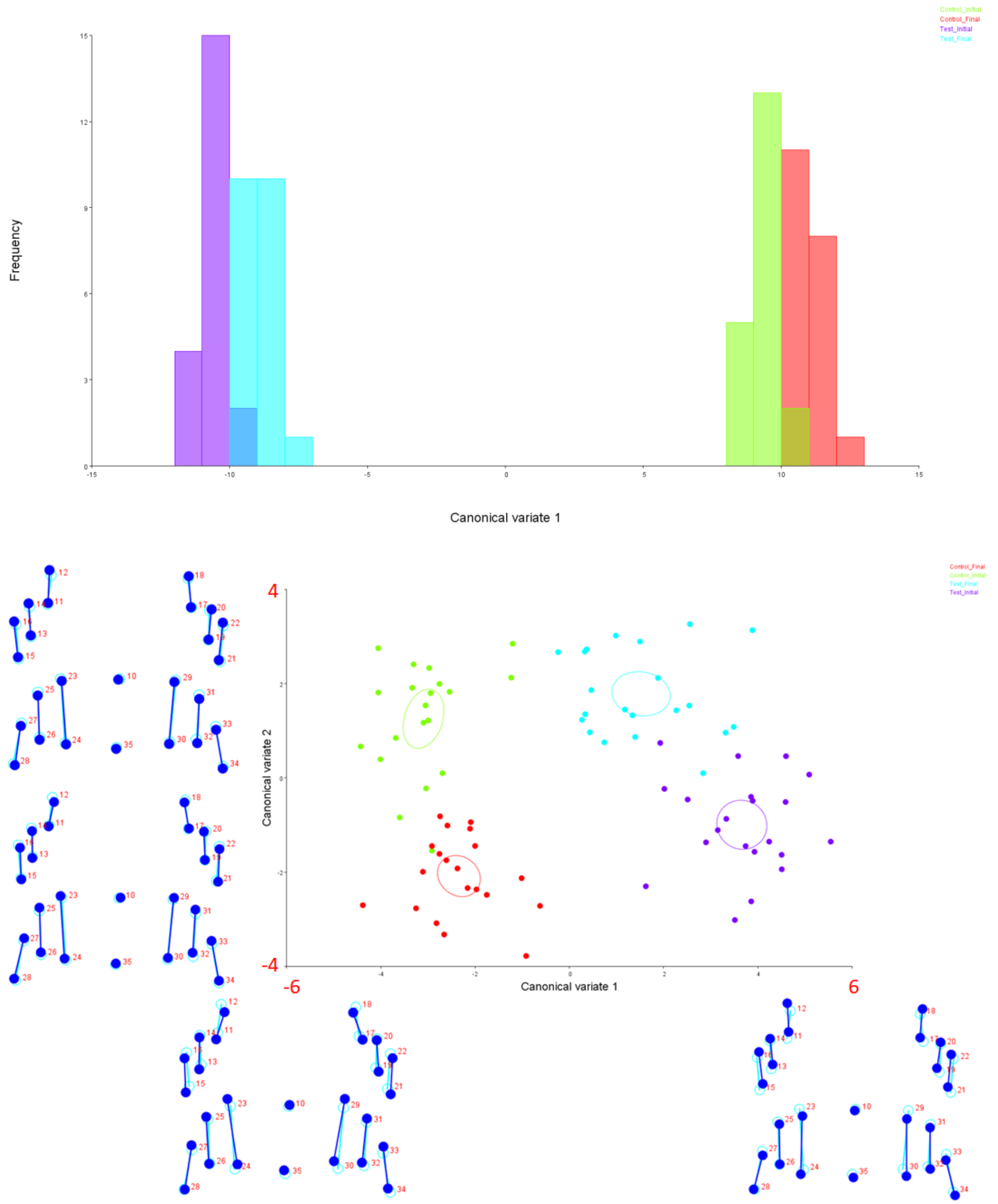



***Figure 16. Canonical Variate Analysis with Age Regression: Initial and Final Timepoints***
*When given the groups identifiers, the canonical variate test clearly distinguished between control and test groups. This artificially maximized the differences present. The landmark graphs illustrate that there were only minor shape differences between the groups. These differences observed were statistically significant (p=0.0365).*

A canonical variate analysis was performed with a regression for age that controlled for treatment time. This analysis was used to determine if there were any identifiable differences between treatment groups and timepoints. Unlike the principal component analysis, this analysis was given group identifiers outright, allowing it to maximize any differences present. Figure 16 illustrates the shape differences between groups. The test group (blue, purple) is separated from the control group (red, green) along canonical variate 1. This separation is statistically significant ($p=0.03$) and represents a clear shape difference between the control and treatment groups that is illustrated in the landmark graphs along the x-axis. These shape differences were very subtle, but the upper and lower canines and premolars of the control group (with more negative CV1 scale factors) demonstrated more buccal flaring, whereas in the treatment group (with more positive scale factors) they were more upright. This landmark graph reinforced the CBCT findings in figure 13 and the principal component findings in figure 12.

This geometric morphometric analysis illustrated subtle angulation differences between treatment groups. Patients treated with phase I expansion had canines and premolars that appear slightly more upright than those who were untreated.

**Discussion**

This study found evidence that the eruption vector of the canines and premolars in patients who had phase I expansion in the early mixed dentition was slightly more upright compared to patients who had no treatment.

A principal component analysis at the initial timepoint showed that no significant shape differences existed between groups (p=0.5976). There were slight differences observed between groups when comparing the shape changes across principal component 5 and principal component 7, as demonstrated in figure 9, however these differences were not significant and were only responsible for 7% and 4% of the shape variation respectively. It is unlikely that this non-significant difference between minor principal components had a substantial influence on the outcome of the study. Principal components 1 and 2, together describing 33% of the shape variation in the sample, showed no differences (figure 8), and there were also no principal components other than 5 and 7 that showed shape differences.

Comparing the initial and final timepoints using a modified Pythagorean theorem, there appeared to be a pattern of angular changes associated with early phase I treatment. This pattern was most clearly demonstrated in the lower arch, with the control group showing less of an angular change than the treatment group. This trend supported our hypothesis that phase I treatment would influence the angle at which the permanent teeth erupt. The angular changes in the upper arch were inconsistent, although the upper first premolar followed the same trend as the lower arch. The upper second premolar showed no differences between the test group and the control group. There are a few reasons why the upper second premolar may not have been affected. Firstly, the upper primary molar is a very large precursor, consistently maintaining extra space for the erupting premolar. Perhaps this tooth is more insulated from the space created with expansion, whereas other teeth without this buffer may upright more when given more space during eruption. Also, the posterior and superior regions of the maxilla are least expanded with RPE because of the pyramidal nature of midpalatal suture separation. The upper second premolar is the farthest posterior in this study and, at the time of expansion, the farthest superior, so perhaps there is a diminished skeletal effect surrounding this tooth. The upper canine showed the opposite trend from the other teeth, however this tooth is typically more susceptible to anteroposterior than transverse malocclusions, so it is not entirely surprising that this tooth did not demonstrate the same transverse pattern as the premolars. This analysis supported the likelihood that there may be differences in the angulation of the premolars in patients who have had phase I expansion compared to patients who have had no treatment. Because none of these differences were statistically significant, however, it is impossible to make any conclusions.

Shape differences were evaluated at the final time point using a principal component analysis to determine whether the ultimate angulation of the teeth was different between the treatment group and control group. There were differences between principal component 2 and principal component 8, as seen in figure 12. Principal component 2 also showed slight, non-significant differences with several other principal components. As principal component 2 increased, there was dramatic palatal uprighting of the upper canines and premolars and mild buccal uprighting of the lower premolars. This is typically associated with normal eruption as the lower teeth erupt lingually and the upper teeth erupt buccally and they both upright into occlusion. Our hypothesis was that this uprighting would be seen more in the test group compared to the control group, which was what was demonstrated in this analysis. In figure 12, the control group clustered towards the low to negative end of principal component 2's axis, where there was a greater angulation of all of the teeth. The treatment group clustered towards the higher values of principal component 2, where the upper and lower teeth became more upright. Figure 13 reinforced these findings, by stitching together PC2 with a CBCT image to precisely illustrate how in the control group, the upper teeth have a much more dramatic buccal inclination than in the treatment group.

We considered the possibility that the angulation of the teeth during eruption may be associated with factors other than expansion. We found that the amount of mandibular crowding, overbite, mandibular plane angle, and age that phase I was started had no influence on the eruption angulation, both at the initial and final timepoints.

The canonical variate analysis shown in figure 16 was given all of the different treatment groups and timepoints to maximize intergroup differences. This analysis demonstrated statistically significant shape differences between the control group and the treatment group, however the shape differences observed were minor. This reinforced the angulation changes observed in our Pythagorean theorem analysis and principal component analysis.

Our power analysis indicated that a sample size of 54 with 27 in each treatment group was needed for a paired analysis. Due to the constraints of a retrospective clinical study, we were unable to achieve the sample size needed. Despite this, we found statistically significant differences between groups in our canonical variate analysis. This analysis maximizes intergroup differences, which offset our underpowered sample. A principal component analysis observed differences across principal component 2 that were nearly significant (p=0.07) and may have been significant with a larger sample size. Future studies would benefit from increasing the sample size to evaluate these differences and may also consider measuring the buccal bone width around the canines and premolars to evaluate whether the minor angulation changes we found could have more meaningful implications for the periodontal health of the developing teeth.

Geometric morphometric analysis was an effective tool to measure angular changes in the erupting teeth by evaluating the overall shape changes in the dental arch. In the principal component analysis of the sample observed at the final time point, principal component 2 was responsible for 14% of shape variation and showed differences between the control group and treatment group that were not statistically significant. Our canonical variate analysis across the complete sample demonstrated minor shape differences between the control and treatment groups that were statistically significant (p=0.03). This supported our hypothesis that the canines and premolars were slightly more upright in patients who had phase I expansion than patients who did not.

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